

# EXPERIMENTAL SETUP FOR THE QUANTIFICATION OF THE CARDIO-PULMONARY RESPONSES TO DIFFERENT ARTIFICIAL RESPIRATORY LOADS

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**Abstract**—In order to investigate the effects induced on respiratory and cardio-circulatory activity by mechanical dysfunctions in the respiratory system, devices were designed and constructed for increasing both resistive and elastic respiratory loads, thus simulating obstructive and restrictive pathologies, respectively. The resistive load was obtained as a duct, with a resistive core, having linear characteristics; elastic load was obtained by imposing the respiration from a closed volume. The elastic load too had linear characteristics. The artificial loads were calibrated for producing a total load about three times the normal intrinsic loads and were applied to a group of normal subjects. A set of signals from the respiratory and cardio-circulatory system were recorded and analysed through proper algorithms for obtaining quantitative parameters both in time and frequency domain.

**Keywords** - Cardio-pulmonary interaction, Heart Rate Variability, Arterial Pressure Variability, Artificial Respiratory Load

## I. INTRODUCTION

Most of the pathologies affecting the respiratory system are characterised by altered respiratory mechanics. Patients affected by *obstructive lung disease* have a reduction in airway calibre determining an increased airflow resistance. On the other hand, subjects with *restrictive diseases*, show an increased elastic recoil at any given volume due to either lung or chest wall disorders. From a mechanical point of view these two *deficit* can be modelled by increasing the resistive and elastic loads, respectively.

The adaptation to these abnormalities in respiratory mechanics are partially known for the respiratory system, while the effects on the cardio-circulatory system in the short, as well as in the long-term period, are to a large extent still unknown.

Aim of the present study is a better comprehension of the effects induced on the cardio-circulatory and respiratory systems by altering the mechanical load imposed on the respiratory muscles.

The effects on respiration are evaluated through the analysis of the air flow signal. The effects on the cardio-circulatory system are studied through the analysis of the beat-to-beat variability signals: RR series, extracted from the ECG, beat-to-beat systolic arterial pressure (ABP series) from the aortic blood pressure and respiratory series obtained by sampling the Flow signal in correspondence of each R peak. Both RR and SAP are affected by the respiratory activity through different mechanisms, both neural and mechanical [1-3]. Spectral and cross-spectral parameters are indices able

to quantify the action of the increased respiratory loads on the system controlling their variability.

In order to 1) exclude the effects induced by other alterations not strictly related to the increased respiratory load, but rather due to comorbidities, 2) have a direct comparison with normal conditions, artificial loads, resistive and elastic, were applied to young normal subjects.

## II. METHODOLOGY

### A. Reference model

Different models have been proposed in the literature for the mechanical characterization of the respiratory system. One of the most popular was presented in [4] and describes the muscle inflow pressure ( $P_{mus}(t)$ ), needed for causing the airflow, according to the following expression:

$$P_{mus}(t) = E_i \cdot V(t) + R_i \cdot \dot{V}(t) \quad (1)$$

where  $V(t)$  is the inspired air volume and its first derivative  $\dot{V}(t)$  is the air flow.  $R_i$  and  $E_i$  are the system intrinsic resistance and elastance, respectively. According to (1), the relation between muscular strength and air volume and flow is linear: in fact  $R_i$  and  $E_i$  values are constant for normal subjects (analysed in the present study), whose respiratory rate is under 2 Hz [5].

Mechanical dysfunctions are accounted for in the model through an incremental load, resistive ( $R_L$ ), for obstructive diseases, or elastic ( $E_L$ ), for the restrictive ones:

$$P_{mus}(t) = (E_i + E_L) \cdot V(t) + (R_i + R_L) \cdot \dot{V}(t) \quad (2)$$

The equation evidences how the adjunctive loads require an additional muscle strength for obtaining the same air volume and flow. In the following a description is given of the devices designed for producing the additional loads and thus simulating the pathology.

### B. Resistive load

The air flowing inside a tube is modelled as a fluid. An adjunctive resistance is produced by inserting in the pipe an obstructive device (in our case a packet of grids) able to cause a pressure decrease,  $\Delta P(t)$ , that is function of the instantaneous flow  $\dot{V}(t)$ :

$$\Delta P(t) = R_L(t) \cdot \dot{V}(t) \quad (3).$$

## Report Documentation Page

<b>Report Date</b> 25OCT2001	<b>Report Type</b> N/A	<b>Dates Covered (from... to)</b> -
<b>Title and Subtitle</b> Experimental Setup for the Quantification of the Cardiopulmonary Responses to Different Artificial Respiratory Loads	<b>Contract Number</b>	
	<b>Grant Number</b>	
	<b>Program Element Number</b>	
<b>Author(s)</b>	<b>Project Number</b>	
	<b>Task Number</b>	
	<b>Work Unit Number</b>	
<b>Performing Organization Name(s) and Address(es)</b> Department of Biomedical Engineering, Polytechnic of Milano, Italy	<b>Performing Organization Report Number</b>	
<b>Sponsoring/Monitoring Agency Name(s) and Address(es)</b> US Army Research, Development & Standardization Group (UK) PSC 802 Box 15 FPO AE 09499-1500	<b>Sponsor/Monitor's Acronym(s)</b>	
	<b>Sponsor/Monitor's Report Number(s)</b>	
<b>Distribution/Availability Statement</b> Approved for public release, distribution unlimited		
<b>Supplementary Notes</b> Papers from the 23rd Annual International Conference of the IEEE Engineering in Medicine and Biology Society, October 25-28, 2001, held in Istanbul, Turkey. See also ADM001351 for entire conference on cd-rom.		
<b>Abstract</b>		
<b>Subject Terms</b>		
<b>Report Classification</b> unclassified	<b>Classification of this page</b> unclassified	
<b>Classification of Abstract</b> unclassified	<b>Limitation of Abstract</b> UU	
<b>Number of Pages</b> 4		

In case of laminar flow,  $R_L(t)$  is constant according to the Hagen-Poiseuille law. However, in the present case, the air flow is non-laminar, thus the resistive element would be non-linear function of the flow intensity. The flow can be made close to laminar by placing two other grids at a suitable distance before and after the resistive element. Fig.1a shows the scheme of the resistive load and the grid system, and the qualitative flow motion in the different sections. The  $R_L$  value is determined by the number  $n$  of the grids inserted in the core. The value of  $n=4$  was experimentally determined in order to obtain an adjunctive artificial load about twice the total resistive normal load. Fig.1b shows device the characteristic curves with (1) and without (2) the regularizing grids. In presence of the regularizing grids the curve is very close to linearity.

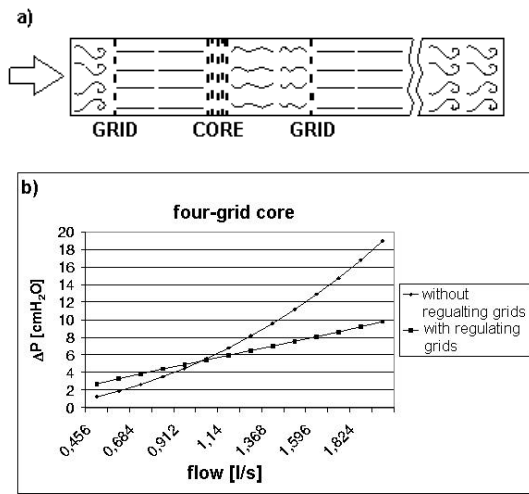


Fig.1 a) Scheme of the resistive core with the grid system used for making the flow laminar. The qualitative flow motion is shown in the different sections. b) Characteristic curves of the resistive element with (1) and without (2) the regularizing grids.

### C. Elastic load

An elastic load produces a pressure variation  $\Delta P(t)$ , that is function of the inspired air volume  $V(t)$  and that contrasts the inspiration pressure:

$$\Delta P(t) = -E_L(t) \cdot V(t) \quad (4)$$

The above load is obtained if the subject extracts the inspired air volume from a closed volume  $V$ . Fig.2a shows the elastic load inserted in the respiratory circuit. The three way valve connects the elastic load during inspiration (Fig. 2a), while expiration is free (Fig. 2b). The electro-valve is opened during expiration (Fig. 2b), when the load is disconnected from the subject airways and permits to restore atmospheric pressure in the load volume. The elastic load was constructed as a plexiglass box, with the described valves, and partially filled of water in order to choose the closed volume  $V$ , thus calibrating the applied load  $E_L$ . The value of  $E_L$  was chosen as 10 cmH<sub>2</sub>O/l, about twice the intrinsic load  $E_i$ . The calibration curve for such a value of  $E_L$

is shown in Fig.2c, and presents good characteristics of linearity.

### D. Experimental protocol

The study involved 5 young (age:  $26.8 \pm 5.3$ , mean  $\pm$  s.d.) normal subjects, without history of respiratory or cardiovascular pathologies. They were lying on a bed in half-sitting position, and the signal reported in Tab.1 were recorded through the listed devices.

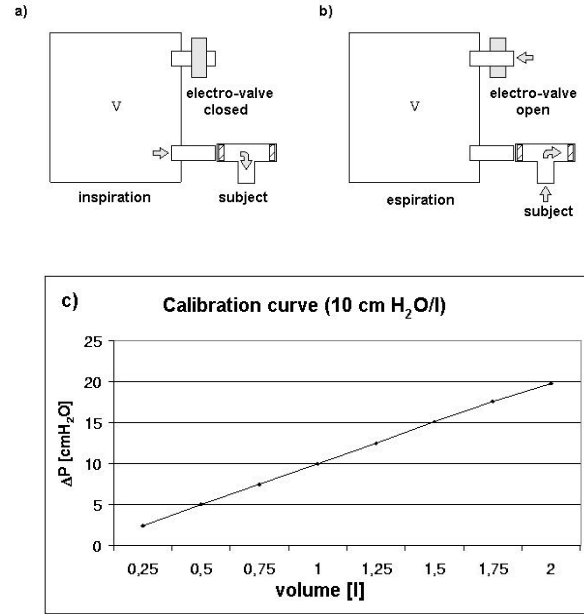


Fig.2 Elastic load with the air-flows through the valves, during the inspiration (a) and the expiration (b) phases; c) characteristic curve for the chosen load value.

TABLE I  
RECORDED SIGNALS

Channel	Signal	Device
$P_{res}$	Pressure after resistive load	Differential pressure transducer, SC_PCB, Sensortech, USA
$P_{el}$	Pressure inside elastic load	Differential pressure transducer, SC_PCB, Sensortech, USA
$P_{aw}$	Air way pressure	Differential pressure transducer, SC_PCB, Sensortech, USA
Flow	Respiratory flow	Pneumotachograph, 3700B Hans-Rudolph, USA, and differential pressure transducer, CD15, Valydine, USA
ECG	ECG (V2 lead)	ECG monitor Seris 900, Marquette, USA
ABP	Arterial blood pressure	Finapres 2300, Ohmeda USA
$SpO_2$	Oxygen saturation (%)	Saturimeter, Oxicap 4700, Ohmeda, USA
$EtCO_2$	$CO_2$ partial pressure (expiration end)	Gas Analyzer, Oxicap 4700, USA

Each subject underwent four recording sessions, 10 min each, under different loading conditions: B (baseline), no load was applied; E (elastic load); R (resistive load), C (combined load, both resistive and elastic loads applied together). The sequence of the epochs was randomised in order to eliminate

a possible dependence of the results on the application sequence.

The analogical signals were A/D converted at 300 Hz sampling rate, 12 bit resolution, using an A/D board PCI-MIO-16E, National Instruments, USA, and stored on a PC hard disk.

### III. RESULTS

#### A. Time domain analysis of respiratory patterns

The Flow signal was segmented, through a zero-crossing procedure and a slope analysis, in single respiratory cycles (conventionally each respiration starts at the beginning of the inspiration). The beginning of the expiratory phase was also detected. Mean value and standard deviation was calculated for the following parameters: *total respiration time* ( $T_{tot}$ ); *inspiratory* and *expiratory time* ( $T_i$  and  $T_e$ ); *duty cycle* ( $T_i/T_{tot}$ ) and *variation coefficient* ( $CV\cdot T_{tot}$ ). After integrating the Flow signal the cycle-by-cycle *tidal volume* ( $V_t$ ) was obtained.

Fig.3 shows the mean total respiration time (in seconds) for each subject during the different recording sessions.

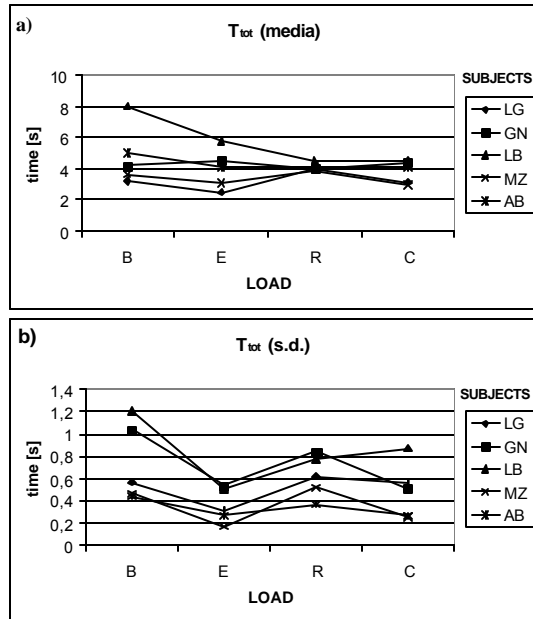


Fig.3 a) Mean value of the total respiratory time for each subject during the different loading conditions; b) cycle-to-cycle standard deviation of the total respiratory time for each subject during the different loading conditions

The effects induced by the different respiratory loads on the total respiratory time (Fig.3), depends on the load itself. R load do not modify the intra-subject breath-to-breath variability: in fact the standard deviation shown in Fig.3b is unchanged, but seems to concentrate the inter-subject mean respiration time around a common value. On the contrary, E load causes a decrease in the respiratory period (Fig.3a), with significant decrease in breath-to-breath variability as shown in Fig.3b. The C load produces an intermediate behavior. Similar trends were found for the inspiration and expiration time and for the current volume.

The reason of the different behaviour could be found in the different nature of the loads. In fact the R load, is a flow-function, acting during the whole inspiratory phase, while the

E load is volume dependent and acts mainly at the end of the inspiration, when the volume reaches its maximum, thus reducing breath-to-breath variability.

#### B. Frequency analysis of the cardiovascular variability

The RR, SAP and respiratory series were analysed in the frequency domain through AR models both in the mono and in the bivariate form, in order to obtain spectral and cross-spectral parameters able to put into evidence variation induced in the cardio-circulatory interactions. In particular LF and HF power and frequency have been related to the autonomic control of the RR variability [1], while coherence function and phase relationships describes the mutual relationships and influences between couples of signals [2].

Fig.4 displays the HF power expressed in normalized units, i.e. as a percentage value on the total spectral power without the very low frequency component [1]. PHFnu shows an increased value in correspondence with the E load and low variation or a decreasing trend in correspondence of the R load. The result is in accordance with the decreased variability in the  $T_{tot}$  parameter in correspondence of E load, that testify an increased regularization of the breath, reflected in a synchronization of the respiratory arrhythmia of the ECG. The phase parameter between RR and respiration series is shown in Fig. 5. It represents the phase delay of the Flow signal with respect to the RR series. A positive value indicates an anticipation of the respiration with respect to RR. In basal condition respiration anticipates RR and the phase is increased during E loading, while increases again in two subjects, decreases in one and remains unchanged in two during R load.

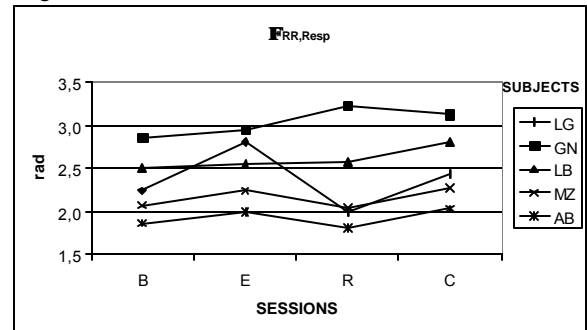


Fig.4 Mean value of the PHF in normalized units for each subject during the different loading sessions

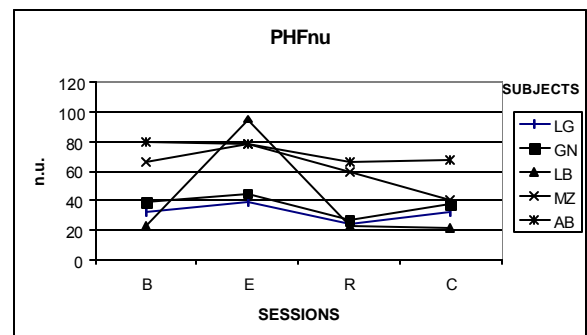


Fig.5 Mean value of the phase between the RR series and the respirogram in normalized units for each subject during the different loading session

The phase delay between RR and SAP series (not shown) is close to zero in B condition and increases in 4 of the 5 subjects with E load (SAP anticipates RR series). An increased delay of RR with respect to SAP may be interpreted as a delayed baroreceptive response.

#### IV. CONCLUSION

The present study was aimed to the the investigation of the effects of different respiratory loads on cardio-pulmonary interaction and variability. The artificial resistive and elastic loads were designed and constructed with linear characteristic and high flexibility for load changes.

The responses to increased resistive (R) and elastic (E) loads were different for both respiratory and cardio-pulmonary systems. Parameters achieved from the analysis of the air flow signal confirmed previous studies reported in literature [6]. E caused a regularization of the breath, testified by decreased  $T_{tot}$  and  $V_i$  variability and by the increased PHFnu of the RR series. R produced the opposite effect (increased  $T_{tot}$  and  $V_i$  variability and decreased PHFnu).

Effects on the cardio-circulatory system were evidenced through the changes in phase relationships between RR and SAP series. The loads caused an anticipation of the SAP maxima with respect to RR, putting into evidence a delayed baroreceptive response.

The above results were common to the whole examined population, however they need a further validation on a larger number of subjects.

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